Aortic dissection originating from an aberrant right subclavian artery

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A 47-year-old male patient presented with aortic dissection originating from an aberrant right subclavian artery. Intraoperative arteriography showed an anomaly of the aortic arch including a common carotid trunk and an aberrant right subclavian artery. An intimal tear was located in the aberrant right subclavian artery. Dissection retrogradely involved the aortic arch and then anterogradely involved the distal aorta. We treated the patient endovascularly with a Wallgraft endoprosthesis deployed in the aberrant right subclavian artery to seal the entry site. Follow-up computed tomography showed a satisfactory healing process with complete thrombosis in the proximal part of the false lumen. (J Vasc Surg 2007;46:1270-3.)

Aortic dissection is a common disease with a high mortality rate that is characterized by the separation of aortic wall layers by extraluminal blood.[1] Intimal tears are most likely located in the ascending aorta and in the first portion of the descending aorta, but primary entry sites in the aortic arch or the abdominal aorta are also frequently observed in clinical practice.[2-4] We have recently treated a rare case with aortic dissection originating from an aberrant right subclavian artery (ARSA) with endovascular therapy. As far as we can determine, this may be the first reported occurrence of aortic dissection that originates retrogradely from an ARSA.

CASE REPORT

A 47-year-old man was admitted to our hospital with intermittent chest pain that had been present for 75 days. Shortly after the sudden onset of pain in the chest and upper back, aortic dissection was detected with abdominal color Doppler ultrasonography and contrast-enhanced computed tomography (CT; Fig 1, *A*) at a local hospital. After medical therapy at the local hospital, the acute chest pain had been relieved, but the intermittent chest pain continued. The patient was otherwise healthy, with no history of hypertension, coronary artery disease, diabetes mellitus, or hyperlipidemia. We applied the necessary treatment, including antihypertensive therapy and analgesics. After necessary laboratory tests and thorough consideration, we decided to treat the patient with endovascular therapy.

In the operating room, we administered general anesthesia via tracheal intubation. Arteriography of the entire aorta through the left brachial access was performed with full consideration of the important branches of the aorta. We observed two arterial anomalies: a common carotid trunk that branched into two common carotid arteries and an ARSA arising from the aortic arch posterolateral to the origin of the left subclavian artery (Fig 2, *A*). Entry sites were discerned in the ARSA and the aorta near the origin of the celiac artery (Fig 2, *D*). The intimal tear in the ARSA seemed to be responsible for most of the blood inflow into the false lumen. The false lumen had been enlarged to form a dissecting aneurysm

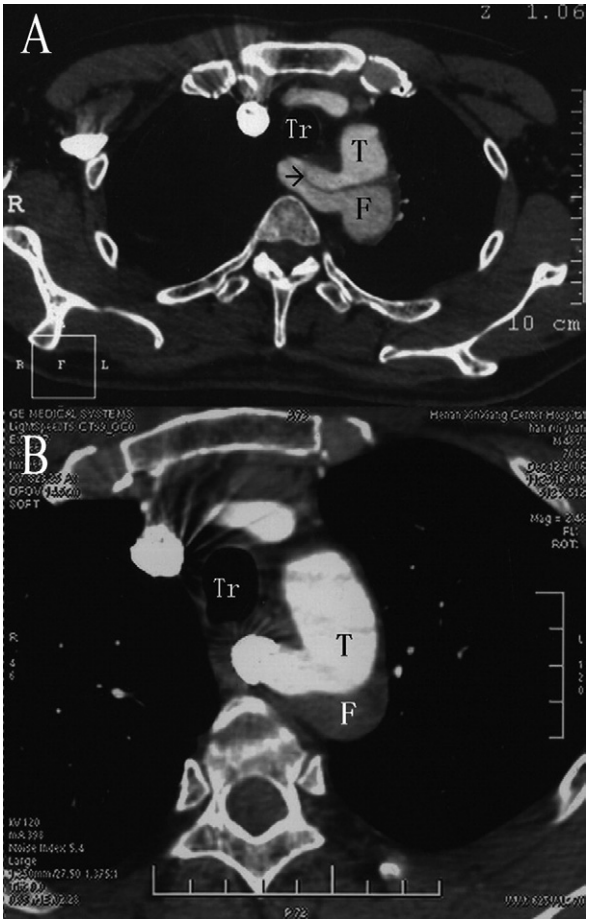

Fig 1. Contrast-enhanced computed tomography (CT) demonstrated the aberrant right subclavian artery dorsal to the esophagus and trachea *(Tr)*. **A,** Preoperative CT showing the dissection *(arrow)*, the true lumen *(T)*, and the false lumen *(F)*. **B,** Follow-up CT showing the thrombosed false lumen *(F)*.

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Competition of interest: none.

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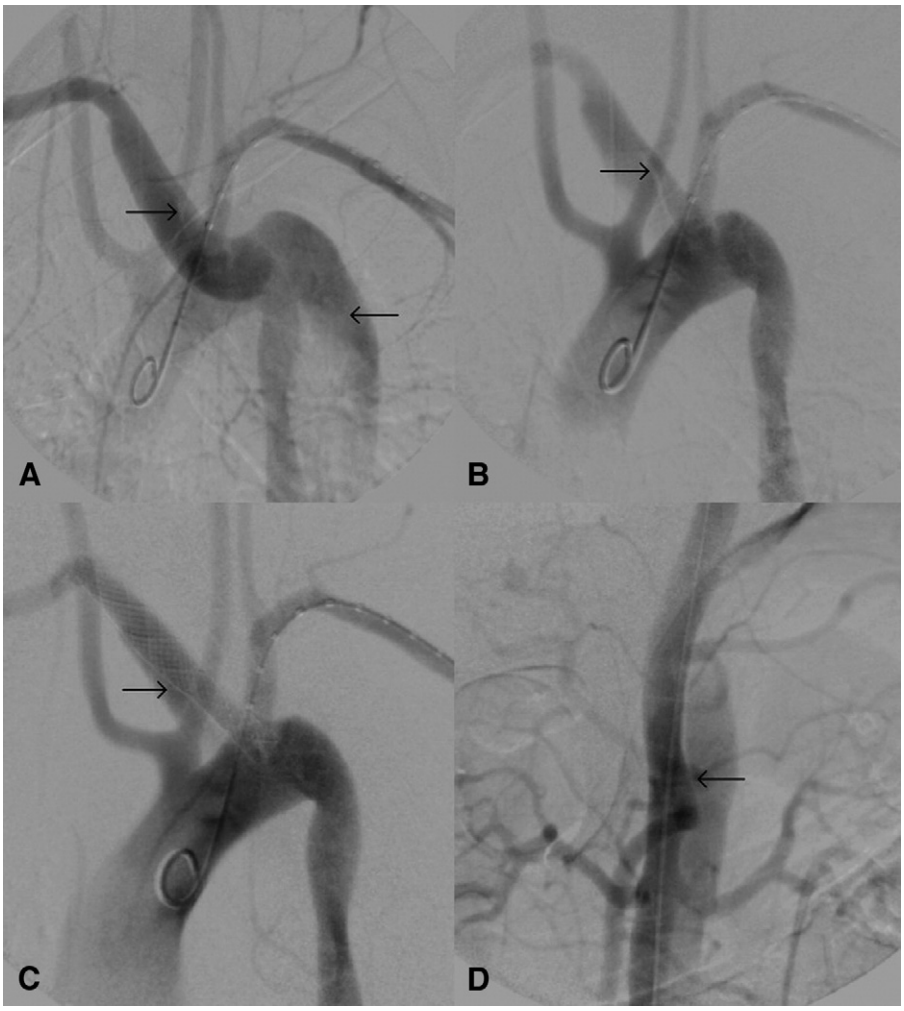

Fig 2. Intraoperative arteriograms. **A,** The anomaly of the aortic arch, the intimal tear in the aberrant right subclavian artery (ARSA), the dissection, and the proximal false lumen. **B,** The true lumen of the ARSA was partly compressed *(arrow)*. **C,** A type IV endoleak *(arrow)* and a diminished false lumen after endovascular treatment. **D,** The entry site *(arrow)* in the abdominal aorta near the celiac artery.

(Fig 2, *A*). Furthermore, we selectively advanced the calibrated pigtail catheter into the right subclavian artery through the left common femoral artery access for a more precise measurement.

We decided to seal the entry site in the ARSA with Wallgraft endoprosthesis (Boston Scientific Inc, Natick, Mass). The right common femoral artery was exposed via a right inguinal longitudinal incision. After general heparinization, arteriotomy was performed, and a stent graft (diameter, 12 mm; length, 50 mm) was advanced into the right subclavian artery over a superstiff wire. With deliberate positioning, we released the graft at an appropriate position. Intraoperative arteriography showed that the graft placement was optimal and that a minor type IV endoleak was present (Fig 2, *C*). We finished the operation and decided to monitor the endoleak during the follow-up period.

The postoperative course was uneventful, and the patient was discharged on the seventh postoperative day. The intermittent chest pain was relieved after the operation. Follow-up contrast-enhanced CT (3 months after the operation) showed a thrombosed false lumen from the ARSA to the descending aorta (Fig 1, *B*, and Fig 3, *A*). The endoleak was undetectable, and the graft did not migrate. The false lumen of the abdominal aorta was still present, but no further enlargement was noticed (Fig 3, *A*).

DISCUSSION

An aberrant subclavian artery, usually the right, is a relatively common anomaly of the aortic arch, with a reported incidence of 0.5% to 1%,[5] and is most often asymptomatic unless a compression effect or atheromatous disease involvement occurs.[6-8] The aberrant subclavian artery may also be involved in aortic dissection,[9,10] as seen in this report.

The anomaly of this patient was not discovered until contrast-enhanced CT was performed for the acute chest

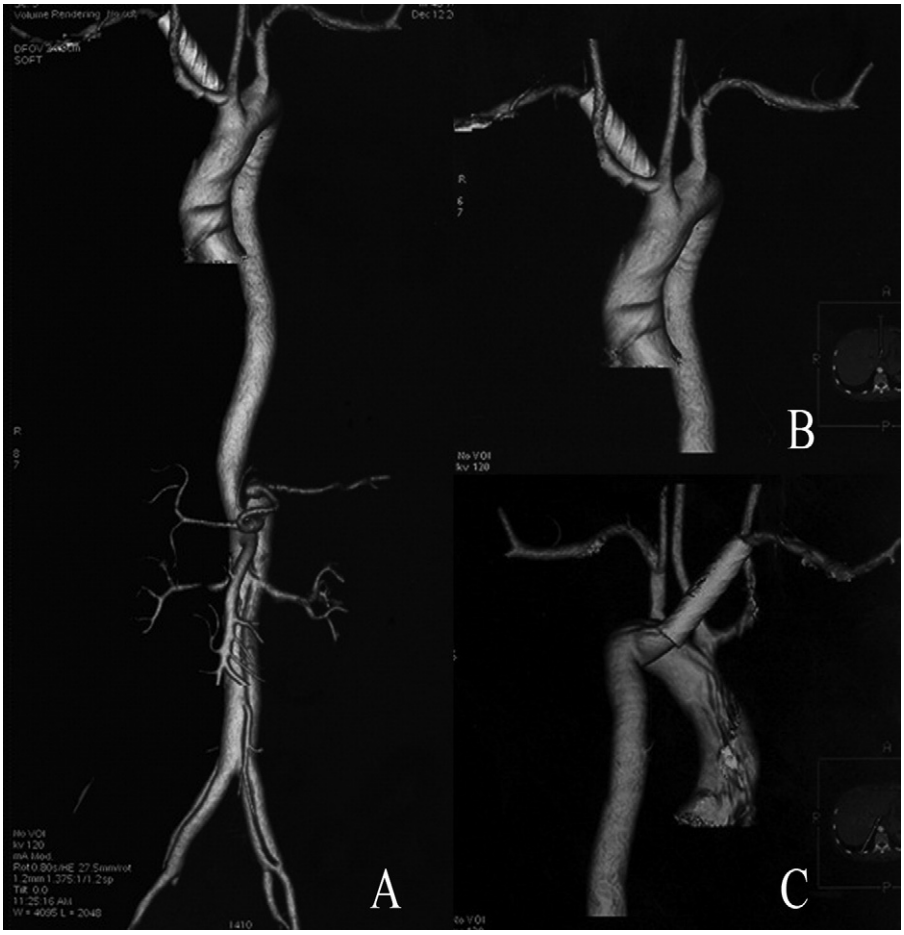

Fig 3. Follow-up computed tomography (CT) reconstruction (3 months after the operation). **A,** Total aortic CT reconstruction. Note the aortic dissection in the abdominal aorta and bilateral common iliac arteries. **B,** Anterior aspect of the aortic arch. **C,** Posterior aspect of the aortic arch. Note the origin of the aberrant right subclavian artery.

pain. Although the ARSA crosses the midline dorsal to the esophagus and trachea (Fig 1), symptoms due to compression have not been reported in this patient. Possible compressing effects of the stent graft warrant further investigation. During the intraoperative angiography, blood inflow was larger through the entry tear in the ARSA compared with that through the entry site in the abdominal aorta (Fig 2, *D*). Therefore, we considered the entry site in the ARSA to be the primary one and the other in the abdominal aorta near the celiac artery to be secondary. Furthermore, the satisfactory healing process after the sealing of the entry site in the ARSA provides convincing evidence that the intimal tear and the subsequent dissection of the ARSA retrogradely involved the aortic arch. Although aortic dissection involving the aortic branches and retrograde dissection are common in clinical practice,[11,12] dissection originating retrogradely from a branch vessel is relatively rare.

Medical therapy remains the first choice for uncomplicated type B aortic dissection.[11] Besides surgical aortic replacement, endovascular therapy with stent grafts has been accepted as a choice for selected patients with complications, although the long-term benefit still needs to be established.[2,13-15] Intraoperative angiography demonstrated that the proximal false lumen had developed to a dissecting aneurysm and that the true lumen of the ARSA was partly compressed, although no symptom had presented (Fig 2, *A* and *B*). The intermittent chest pain was considered as a sign of possible further expansion. We chose a Wallgraft endoprosthesis to seal the primary entry site because no other peripheral stent graft was available in our hospital at that time. Moreover, the delivery of the selected stent graft (diameter, 12 mm; nearly 20% oversized) required an 11F introducer, and we preferred femoral access to brachial access. The secondary entry site remained to allow follow-up observation because of its location near the visceral arteries, and there was no occurrence of related compression or ischemic symptoms. After treatment, the intermittent chest pain has not recurred during the follow-up period. Follow-up CT showed complete thrombosis in the proximal part of the false lumen and no signs of residual leakage, but the false lumen of the abdominal aorta remained because of the second entry site (Fig 3, *A*).

Because the false lumen of the abdominal aorta did not enlarge, we chose observation rather than intervention. If further enlargement or persistent presentation of the false lumen occurs, interventions—including endovascular therapy with a fenestrated stent graft or open surgery—may be necessary.

Aortic dissection originating from an ARSA has a very low incidence, and endovascular therapy may be advisable for selected cases, but the long-term outcome still needs to be determined.

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